

**read transcript of 40min mp3 Dr Chetty interview Clinical Implications of
Weaponised Bacteria Against the Host**

Hi. Good evening to everyone. This is a presentation that I wanted to do yesterday, but we had some technical difficulties with doctor Shetty. And, for those who don't know me, I'm doctor Philip McMillan, based in the UK. I've been focused on COVID nineteen from an autoimmune perspective.

And one of the tremendous pleasures I've had over the many years is talking with doctor Shankara Shetty about his research and critically his interventions, from a South African perspective to save the lives of all his patients. Now I think it's very important to listen to what he has to say. And today, we're gonna be focused on the potential impact of weaponization of bacteria. So we're just usually just going to chat about what some of the thoughts that we have. And so without any further ado, with a little bit of flash, let's bring in doctor Shetty.

Great. Shankara, how are you? I think Hi, Philip. Nice to have you. And, welcome to all the people watching us tonight.

Excellent. So, listen, Shankara. As I said, yesterday, we were planning to have this discussion, and then we we had some technical difficulties. I started to speak about, this this recent Carla Brockner paper, which we will go into in a little bit. But before we go into that, Shankara, I just want you to remind the people again, part of the reason why we're speaking, regularly with Shakara is that his license to practice medicine is under threat for saving the lives of his patients as remarkable as it may seem.

I want you, Shankara, to tell us a little bit about this paper that you had done in twenty twenty. What was this about? Philip, I knew when the pandemic arrived, transition from one to, to Italy, that there was a nefarious agenda at play. I had seen the insert in the genome of the virus and knew that it was likely lab made, and so I had to see patients. I had to understand the clinical presentation of how patients progressed into severe illness for me to try and negate that with the treatment strategy.

So I looked at basically all the information that was available around the world, how people were dying, the clinical observations that were being made, the understanding of the virus being a miss an RNA virus, how that usually did did I I looked into the knowledge that we had already and looked at what was on the table and tried to figure out what we were dealing with. It was the reason I decided to see patients to understand

clinical presentation and from that understand why people did the pathophysiology around the illness itself. And very early on in the pandemic, when I started seeing patients, I understood the biphasic nature. I understood that there was an immune dysregulation occurring at a point in time, and that was the critical point to address this. I also understood that that immune dysregulation might be tied in to the insert into this virus.

In that, it was something new. And, of course, we've been exposed to coronaviruses before, and we never have this kind of reaction. So I just looked at all the information around what was known, what was unknown, and tried to formulate a reasonable hypothesis of how to treat this. When I got the treatment successes, my staff came to me and said, look. You gotta share this with the world.

You're getting amazing results. Patients were recovering within a day overnight. And so I had decided I need to publish an academic paper, more an observational and clinical academic tour of COVID, the illness, the pathophysiology, the treatment strategies that should be employed. And of course at that time I had seen about two hundred patients. This article was written in, May, June twenty twenty and got published in September twenty twenty in an academic journal here in South Africa.

So basically, I just wanted to direct early treatment and understanding of what we were dealing with and, of course, to direct research from a pathophysiological perspective so that we could understand, what we need to research in future to get a better understanding of how to more effectively treat this. I didn't expect that to morph into a commentary about pending, medical interventions that might be compromised by my work. Yeah. This this is, it's fascinating. Interestingly, I put the the link to it.

It's a citation, from the people. But there you can't you'd have to probably go to the publication to read it unless you have the PDF. I didn't have a link for the PDF on it. But in it, you know, Shankara, it it's quite clear. And strangely, as hard as it is for the academic community to to acknowledge, you are spot on because you had observed the patients at that time.

You were seeing as as I'm just gonna show it here again. You were seeing because this was, as you said, by May, you had identified the unusual symptoms with the hypoxemia, the sudden onset, sudden rapidly progressive, breathing difficulties. You know, they drop in the saturations. You're looking at the autopsy findings with heavy edematous lungs and

the microclots. Critically, these microclots, the multiple organ involvement, disseminated intra intravascular, complications.

You're looking at some of the chronic manifestations that could occur because of it and unusual outcomes. This this was quite profound at the time. I'm I'm I'm telling you, you know, is that it's fascinating to think that the clinical and the scientific community would find criticism of this really forward thinking document in twenty twenty, May twenty twenty. Just for people to remember, this was in May twenty twenty. This was even before Oxford had realized about dexamethasone.

This is quite fascinating. You were using steroids before Oxford did the recovery trial on steroids, And people would want to punish you for that? This is this is just unbelievable. I think, Philip, that that informed my opinion of the narrative around the pandemic. That exact lack of acknowledgment, lack of accreditation, informed my decisions.

I understood that I found something remarkable, and why wouldn't the world want to listen to it? So the lack of acknowledgment was more telling than any acknowledgment I got. And so I started to look at things in a different light from that point on. I I had the knowledge that we're dealing with a the probability of a lab made pathogen at that point. And when I noticed the, illogic of the agenda, the lack of acknowledgment for the discoveries I'd made, I understood that I was dealing with a far bigger picture.

And I think the last few lines of that article are what made it controversial because I mentioned in that that, if early treatment could negate a lot of the mortality and morbidity in the pandemic, it would make an mRNA based, intervention rush to market wholly unnecessary. And, of course, I did say that the messenger that the mRNA was wholly unnecessary. Even the rushing to market would have been unnecessary because we've found a way to negate the mortality and morbidity and could take our time to develop an appropriate strategy with all the safety and efficacy built into it and confirmed from it. You know, it's interesting because I did find that right at the end of your paper in mid twenty twenty, you made the point that if you if there is a solution like this, you know, we can treat patients, and we don't need to do any other kind of intervention. This is probably what got you into hot water with this, and this is probably why this information anyway, let let to you, Philip, I just wanted to add.

When I wrote that article, I knew that it was going to be controversial. I had seen the paper that had come out about hydroxychloroquine and, you know, there was all this,

false reporting by profound journals that should have been, the mainstream. And so I thought, which peers should I approach? And I approached two friends of mine, one a senior gynecologist and one a cardiothoracic surgeon. He was, the cardiothoracic, was actually in Italy at the time looking at COVID, and I sent them both this article and asked their opinion.

I got a response within an hour to say it's the most immaculate piece of science they've seen, but both advised me to remove that last sentence about the vaccine. And I said, no. It needs to be put there. It needs to be understood in the context of what we're dealing with. And little did I realize that, yeah, it would be the most controversial statement in that paper.

And just so, people can see it, I'm trying to oops. I'm trying to see if I can get it here if I can, so that, people understand why this would have been a problem? So here, I have it in the conclusion here. And so you went and said with the high mortality and morbidity from COVID nineteen, A little above that, Philip. Just above.

Oh, it's here. It is oh, yes. Here we go. Oh, boy. I'm not even sure if I can read this, Shankara.

For those people who can read, you can read what he is saying. Effectively, he picked up the point that I think would have hit the industry, and it would have been unnecessary and unsafe in view of the rush to bring it to market without long term evaluation. Goodness, Shankara. No wonder you're getting into trouble. You you're too you're too close to the target.

What's the truth, Philip? Oh, boy. So, you know, so I it's important for people to understand because, recently, we highlighted about, doctor Kuderoff losing his job, talking about lockdowns at Harvard. Doctor Carr, I don't know if you know about her from Canada, a pediatrician who, was criticizing the government with regards to some of the actions. They have pulled back from their, their targeting of her, but she's still left with a huge legal bill, which is which is very sad, and she's trying to fight to get through that.

But it's just showing you that, sadly, this is pretty, pretty serious. And so this is why I think I'm highlighting now, and we're gonna transition into the bit that I wanted us to talk about. You had pointed out something then, and let me see if I can get it here. Yeah. I've got it here.

So look at this here. In that same paper, you highlighted that gastrointestinal infection is common, usually preceded by a sore throat, then spontaneously results in a day or two. Heartburn, nausea, short, severe intermittent abdominal cramps with severe diarrhea. That's it slows to a poorly formed, sometimes slightly stool in four to five days. So you were noticing this gastrointestinal pattern, and it had made you think about the interaction with the microbiome at that time, didn't it?

Yes. Yes, Philip. It did. As well, I did mention in the articles that the, the Chinese at the time of early investigation had found a positive PCR rectal swab, and I had, asked for consideration of a fecal oral route of transmission. We were only talking about airborne spread.

And if we had a positive PCR rectal swab, then there is viral genome in the stool itself. And if that viral genome is there, is there a possibility of a fecal oral route of spread? And if there was, then you could pick this up from a public toilet without anyone else being in there, and the masking couldn't solve that problem. So it put the public health measures into a little bit of a different light. Subsequently, we it I think a year later, they identified it in sewage, And I still couldn't understand why we weren't looking upstream to, stool samples and trying to understand how this virus was interacting with the cuts and, of course, the microbiome.

Yeah. And so it it leads me into, important point, and I I've I've got this this paper that I'm gonna show you here. This is central to a publication we're working on about, long COVID type patterns. And it's because it's one of the at the time, it was one of the few papers that was looking at biopsies in the gut, and they were looking at viral persist antigen persistence in the intestine. And they took a number of patients with inflammatory bowel disease.

And, effectively, what they were doing so they were doing an endoscopy, forty six patients with inflammatory bowel disease. Two hundred and nineteen days after, a range of up to two hundred and fifty seven days after confirmed infection. And what they were finding is that they were having antigen persistence of many of the critical proteins, nuclear capsid, you know, spike protein, with associated symptoms from the, long COVID type picture. So what it's clearly demonstrating, and this is what we had thought before, is that there is an element of viral long term persistence that was driving symptoms. But it's not until Carla Brockner's work came out that we started to think really properly about that this may be bacterial.

By the way, let me let the audience know something. What we're talking about here is so important and so significant that I wouldn't be surprised that you will struggle to see this discussion. It is such a significant discussion. This is what we're looking at now. If and let's just let's just give the presumption that Carlo maybe wasn't right.

Let's just say if we have virus being able to replicate or even the proteins, the different, proteins being made by bacteria. Because we're talking about two hundred and fifty seven days after infection. It's almost a year. They are still finding the pieces of the virus in the intestine. Yep.

I think it's, Philip. I think there's more to this than meets the eye. Ever since biotechnology advances started occurring in the late nineteen seventies, early eighties, we should have had the regulations in place to make sure they were done for good intentions. That never happened. I don't think people realize that, biotechnology has advanced to the point where it can challenge conventional, weapons in global warfare.

Yeah. And when you look at what they found, they were breaking it down here into the different categories here. So they were able to identify so they could find the RDRP proteins. I'm not even sure what that one is. Certainly spike, nuclear capsid, and envelope proteins were all present, and this is the percentage of patients who had the so it's not just the spike.

It's a whole viral protein. And so the spike certainly is part of it. And this is one of the other things that was being found is that, you're having viral persistence that can be affected even just from the spike, but certainly from the virus. And so when we have and this is now moving into the clinical bit now. One of the problems we've got is that because the disease is mild at the moment so people either have natural immunity or they have hybrid immunity.

Okay? Now my the the research seems to suggest natural mucosal immunity blocks the virus from getting through the mucosal barrier. But we don't get the same protection with hybrid immunity because it can still penetrate the mucosal barrier, but it doesn't necessarily cause severe disease. In the context of bacteria, if you have virus being able to penetrate and get because it doesn't seem as though it can pass the stomach. So it must be getting to the gut from the bloodstream.

That's my thought. And any any thoughts on that? Do you think it can get through the stomach acid? Philip, we have many enteroviruses that actually are able to do that, that

can directly get into your gut and withstand the acidity in the stomach itself. And then, of course, you have people that are on proton pump inhibitors in the rest that change the environment in the in the stomach itself that allows viruses to get through.

I think as well, immunity is half the picture when we look at what's transpiring in the gut and Carlo's work. Carlo showed that when he took the supernatant and inoculated it onto fresh healthy stool and incubated it, he got an exponential increase in both viral titer, and he got an exponential increase in toxicity in that in that sample. So it showed bacteriophage activity and the virus able to replicate in the gut using bacteria as their host. But, also, he it cuts the bacteria in the gut to produce toxins, and immunity doesn't play a part in toxicity. Now the the issue is that, toxins, peptides or toxin like peptides will be degraded by stomach acid.

But But if you could find a way to have them made directly in your colon, then you'll absorb them unchanged. And so the ideal place to implant toxin would be directly into the colon. And if you could recruit the gut microbiome to make that toxin for you, it would be the ideal environment to create toxicity without the interaction of stomach acids and the rest. And I think that is where when you look at what's transpired, it it it creates more, creates more not confusion, but more skepticism about this being for the good with good intention. Yeah.

It it it really and this is why I was saying yesterday, you know, the the building is on fire. And how do you warn people? I need you to leave the building quickly, but don't panic. You know? Yeah.

But whatever you do, don't ignore it. And so I I did an example yesterday, and I don't know if it made any sense. I said, it it's kind of like, for whatever reason, cyanide was found in your tea. You don't know how it got there. It could have fallen from the ceiling.

Somebody could have tripped and it fell in or something. But if you do not consider the possibility that someone deliberately put it in your tea, there's a pretty good chance you're in trouble. And this is where most of the world and this is what I'm trying to say to people. Listen. Whatever your opinion if if your opinion is that this could never happen, you would make a terrible detective.

Yes. I think as well, Philip, we got to look at synchronicities that we see around what's happening. When you look at toxic peptides, in general, toxic peptides influence physiology in a particular way. Toxic peptides either are cytotoxic, They damage tissue

directly, on contact with them. And, of course, we know about the endothelial injuries and that kind of thing that's by protein has is caused.

Toxic peptides also cause damage through hematologic ways. So they are hematoxic, which will either cause coagulopathies or bleeding disorders, and we've seen that as well with, COVID illness. And, of course, the third mechanism by which toxins work is to cause nerve problems, paralyze their prey, and cause nerve damage, and we've seen that with COVID. So when you look at the broad diverse spectrum of clinical presentation and then understand the toxicology around gut microbiome, there seems to be too much of correlation there for it to be discounted. You know, here's an interesting thought that I I had today when I was discussing.

We're seeing a sharp rise in liver disease. And, I recently did a a presentation about this. But as I was reflecting today, I had a number of different things, the fatty liver. Had spoken about the, the autoimmune hepatitis subclinical either from virus or any other spike routine. And I spoke about alcohol as being a toxin to damage the liver.

And I then suddenly remembered there is one other point because when bacteria in the intestine produce toxins, if they penetrate that gastrointestinal barrier, the first place they are going to hit and need detoxification is the liver. Yep. What would happen if we then had chronic low level toxin production from the intestine hitting the liver. It should be like a continuous blow to the liver, which could then accelerate underlying liver disease. I I really should put that in as part of the presentation.

Any thoughts on that? Yeah. You're absolutely right, Philip. Everything absorbed in your gut goes through the liver first to detoxify before distributing whatever it is that you've absorbed through the body itself. It's a protective mechanism.

We found ways past that through sublingual and, cutaneous routes of administration of certain drugs, patches, hormone patches, things like that. By putting it onto your skin or absorbing it through the mucosa in your mouth, you bypass the liver. That's known as first pass metabolism, a common thing in medicine. All drugs go through that. When you take a drug, we gotta know its first pass metabolism because that will tell us how much is actually getting into the bloodstream for biodistribution.

So the liver is vital in detoxing anything that's absorbed from the gut. And so the liver would be the first point of injury with any, toxic peptide being absorbed in the gut. And, you know, another area as we think about the clinical implications, one of the other things

that I've been hearing about, and, initially, I couldn't make sense of it until recently, is that the issue with teeth because, again, much of the mouth is full of lots of bacteria, especially anaerobic bacteria. And they are usually below the gum line, and so they, they don't need oxygen to replicate. If this cohort of bacteria are also weaponized in the sense that they're producing more toxins, they're damaging their, the counters, bacteria because, important thing for people to understand is that bacteria keep control of themselves because there is limited amount of new of nutrients.

And so they battle for the nutrients, so no one group can outgrow the other. However, if you have a situation where one group or two groups of bacteria are able to produce toxins that destroy the other bacteria, suddenly, they are the king of the hill. And so you end up with overgrowth of bacteria, which could then potentially lead to lead to gingival disease, teeth problems, chronic gingivitis. And then what would happen from that is from for people who are susceptible, infective endocarditis. And that's another thing where the bacteria get into the bloodstream and then infect the valves of the heart.

I mean, the the the implications are, like, endless. I think, I think Philippus has been known for a long time. It's just that it's been kept from general mainstream, science and public interest. I've been talking out against the use of wheat as a source of carbohydrate. We weren't meant to eat that as a source of carbohydrates.

I haven't seen a primate eat grass. Neither have I seen a primate milk a cow. So the two commonest foods that we're allergic to are wheat and gluten gluten and milk. And when you look at gluten, wheat products, we can't we don't have the bacteria in our mouth to degrade it. But every morning, we stick some in our mouth, it gets in our teeth.

And what I found was that wheat is the commonest cause of tooth decay. We, wrongfully ascribe it to the eating of refined sugars. But we have the right bacteria and amount to degrade sugar, and sugar does not persist in your mouth. But we don't have the bacteria for wheat. And so the bacteria and the ASC food in our mouth and start to settle there.

And then, like, we say toxins, they make some of the strong strongest acids and enzymes to degrade those cell walls of this dense wheat. And those strong enzymes degrade your teeth as well and damage your enamel. So I've seen kids that don't eat sweets that have very bad teeth. It's the morning breakfast that's causing the problem. I've encouraged my son to brush his teeth after breakfast, and he's never added a a rotten tooth.

So, yeah, I think there's a lot of the microbiome or the microbiology on the planet that we don't understand, and I think it's vitally important for our survival. Sixty percent of the cells that make up you are not you. They are microbiome. So as general practitioners, we've limited ourselves to treating forty percent of the patient by not understanding the sixty percent that's so vital to our survival. We are an ecosystem, and it's a very balanced ecosystem.

You know, as and some people reading or listening to us would think, well, you know, the solution is just, you know, just use antibiotics. But it's not so simple because your antibiotics, whilst they do do some targeting, can quite easily wipe out even more of your good bacteria. And so you may easily go from a bad situation to a worse situation where there's also resistance. There are all kinds of problems that that occur with this. This is not necessarily easy to fix.

No. Not not not at all for them. If you look at the role of the bacteria in let's take your gut. They digest your food. So your gut is like a septic tank.

And when you put in food there, it gets digested enzymatically digested, by these bacteria. So it releases nutrients from your food that you wouldn't, normally be able to digest and absorb. The gut bacteria also process pathogens that get into your gut, and in that way, stimulate immune response to things throughout the body. And as well, they make certain chemicals that we can't make, which we readily absorb and convert into other other chemicals. So serotonin and things like that are all the starting, compounds are made by the bacteria in your gut.

So someone depressed might have a gut microbiome upset that doesn't give them the chemicals they need to make the feel good factors that cause the depression. So I think the the microbiome has been understudied, but it is I think the the bottom line in what we're dealing with will provide us the solutions if we look at it very closely. I think that's where the solution actually lies. Yeah. It's very, very worrying, even for us from a clinical point of view because we know the complexities of this.

This is not straightforward. And if the scientific community is not even considering it, then there is absolutely no chance. Just to give some more context, I've got here, this is an image of from the paper. So this is easily over two hundred proteins scattered around inside the, the intestine. And some of it, this is just zoomed in.

You can see all of these scattered within. So some is in the lumen. Some is in the submucosal region. Similar picture here. This here is in a cell in the, in the lumen, and then you see scatterings in the sub submucosa here.

This is some pretty serious stuff to have this kind of remnant of the virus existing. And as I said, what happens is once it penetrates the mucosal barrier, it can then diffuse. So it's not even just the gut. It will be, any area that could become infected. Prostate could end up with prostatitis with this kind of, inflammation.

It could literally be anywhere that you have the microbiome existing. And so this is what makes it so very, very complex. And I can't see it here. Yeah. Part part of my consideration early on in the pandemic, we had a very distinct start to the second phase of the illness itself.

Like I've mentioned on numerous times, patients on the eighth day suddenly took a turn for the worse, where on the seventh day, they thought they were completely recovered. And, of course, when we have the development of, antibodies, immunoglobulins, it is a process that's progressive. It's not a sudden onset of something. So I looked at that, and I kept considering, is there something more that we're actually dealing with? And then I spoke to.

He's with, a training for the special operatives in the US Department of Defense. And, of course, there are two things. Only two, physiologic or pathophysiologic processes that are that aggressive and that, lead to such rapid decompensation in a patient. And that is an exposure to an allergen that you are highly allergic to or an exposure to a toxin. And those two things were on my mind from the start.

Now when you look at an exposure to a toxin, that is a sudden event. So I kept considering, is this an infection of the microbiome that doesn't really cause any problems due to the infection? But once the microbiome start to produce toxin, then we respond to that. Either way, the treatment is about the same. You're trying to stop the body's response to something, and it's the response to either a toxin or an allergen that kill the host.

And so the treatment intervention's pretty much similar. It's the reason TAO got in touch with me to plant that seed in my head that we might be dealing with the toxicity rather than an allergen. And, of course, I think there's an interplay. Yep. And, you know, one of the things that I remember when I was looking at some of the long COVID research

around the microclotting and so on, and there was very specific abnormalities with the interaction of von Willebrand factor and from and so on.

And I then because of Carla's work, I then did a backward step and said, okay. Is there any toxin that can replicate this kind of picture? And you know what it was? Shigella. Shigella toxin definitely is associated with clotting and increased clotting.

And and suddenly you realize that suppose this kind of bacteria is then producing higher levels. And it it's small amounts over a long period of time that will overwhelm, eventually, almost wear down the system until it causes disease. And so it's a it's like a, oh, you know what it's like in in the second World War? This is a historical reference. In the second World War.

The Germans came up with the butterfly bomb. Okay. What they did is that they dropped them. And in some parts of the UK, the planes would fly over and then they would drop these bombs that were like butterflies. So the children would then later on go out and see them and play with them and bring them home.

But these bombs had a delayed explosion on it, so within twenty four or forty eight hours, they would just explode. They were absolutely frightening. This is almost potentially that kind of really frightening picture, which you'd if you really have to be suspicious even to think about it, much less to be able to try and anticipate it. Yeah. It's like recruiting the gut microbiome to be that butterfly, that would cause secondary problems, and you'd never understand why it's why it was there.

You know, Philip, the the the clinical observation and understanding of symptomatology is vitally important, and that's the reason I did what I did. In the paper that I've that we've just went through, I mentioned a mucoid diarrhea, and that is dysentery. And, generally, infections of the gut present with a very watery diarrhea. But if you have toxicity and colonic inflammation, then you get mucus production. And now it makes sense that we're dealing with toxicity that creates the mucoid diarrhea.

And like you say, she get a, yeah, dysentery. Toxic. Wow. So listen. Just so, people understand because we're gonna wrap up now, Shankara, as we continue to explore these thoughts, I I think that some of what we said there was so almost, what's the word?

So serious. I have a feeling that those words may not necessarily want to be seen. So if you want to see the full discussion, you must follow the link that will be put below, on the Substack because I think I'll leave there's some parts of this that are almost too serious

that, I suspect those who do not wish this kind of conversation to occur would therefore want it to be taken away. So, yes, folks who are with us, thank you. This is a Thursday evening, and so we appreciate the people who are here with us.

But this is some pretty significant clinical stuff. And I still maintain that doctor Shetty has been there from the start observing the clinical patterns. I can't think of anybody better to try and work out the directions that things could go. So, Shankaran, any final words before we leave? I think, a little bit of hope for people, to understand how bacteria and viruses actually cause, morbidity and mortality in their hosts, our immunity is a pretty remarkable thing, and it can take care of almost any virus or bacteria on the planet.

We developed that immunity through exposure in childhood and build up this library of things we can ignore. So I don't think immunity is a problem. We'll we'll be able to tackle any virus or any bacteria thrown at us. However, the mortality and morbidity from these infections is caused by a host response, not by a but but not by the virulence of the pathogen itself. So the virulence of a pathogen is its ability to trigger a serious host response that eventually kills the host.

So if we gain a decent understanding of how to curb these unusual host responses, then we can deal with any pathogen that's thrown at us. If you look at Ebola, you got the western variant and you got the wild type. The western variant does not kill. The wild type does. And its its lethality rests in its ability to trigger a cytokine storm.

So if we can gain an understanding of how cytokine storms are triggered by different pathogens and the treatment necessary to negate those, then we can deal with any pathogen thrown at us. So there is hope for the future. Absolutely agreed. So listen, guys. Remember to stay with us.

We are right at the front of this trying to figure it out. So if you want to join in the research journey, please continue to stay and listen with this kind of work. Have a great evening, everybody. Shankara, if you could just hang fire with me. Thanks, Philip.